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AN OUTBREAK OF ACUTE RHEUMATIC FEVER IN NOVA SCOTIA

Introduction

Acute rheumatic fever has become uncommon among children in North America over the past four decades[1]. This condition has not been documented in any patient at IWK Grace Health Centre in Halifax, Nova Scotia, for over 15 years. During July and August 1997, three children with confirmed or suspected acute rheumatic fever following Group A streptococcal (GAS) infection were seen at the Centre. The rarity of this condition at the present time in Canada prompted this report on our recent findings.

Case Reports

Case 1: On 21 July 1997, a 10-year-old girl from Truro was taken to the emergency department of the local hospital with acute arthritis of both knees and ankles. She had tachycardia and a systolic murmur. An electrocardiogram (ECG) showed a prolonged pulmonic regurgitation (PR) interval. Mitral and aortic valve insufficiency was confirmed by echocardiogram. She had a history of brief fever and abdominal pain 6 weeks earlier; 3 weeks following that, she experienced upper respiratory symptoms. Two weeks before, on 6 July, she was admitted to hospital for arthritis in the right, first, metacarpo-phalangeal joint. Blood culture at that time grew *Staphylococcus hominis*. She was treated with intravenous and oral anti-staphylococcal antibiotics for a presumptive diagnosis of septic arthritis. She subsequently developed migratory arthritis involving both knees, ankles, and her right foot. She was admitted to the IWK Grace Health Centre for further assessment of this problem.

Investigation showed significant elevation of erythrocute sedimentation rate (ESR) (119 mm/hr), antistreptolysin-O test (ASOT) (600 IU/mL, normal < 200), and anti-deoxyribonuclease (DNase) B titres (1/680, normal < 1/170). Throat culture for streptococci was negative. A diagnosis of acute rheumatic fever with carditis was made based on clinical and laboratory criteria. Treatment with aspirin was initiated, and the arthritis improved dramatically within 24 hours. One month later, her ESR, ASOT, and anti-DNaseB gradually decreased, but the echocardiogram still showed presence of mitral and aortic valve insufficiency.

Case 2: A 6-year-old girl from Stewiacke had intermittent sore throat around the middle of July 1997. On 27 July she had a rash thought to be scarlet fever by her doctor. She was treated for 10 days with erythromycin. On 29 July, she complained of pain in her left groin. Subsequently, she developed migratory pain in her left hip and left knee; this was severe enough to prevent her from walking. She was seen in the emergency department of the IWK Grace Health Centre on 5 August because of peeling from hands and feet. Treatment with aspirin was initiated, and the joint pain resolved very quickly. Blood tests showed elevation of ESR (35 mm/hr) and anti-DNaseB antibody (1/680), although ASOT was normal. She returned on 12 August because of recurrent peeling. Repeat blood tests showed further elevation of ESR (62 mm/hr) and anti-DNaseB (1/1,360); ASOT increased from < 200 IU/L to 400 IU/L. She had a soft ejection systolic murmur. ECG and echocardiogram were normal.

Case 3: A 15-year-old girl from Yarmouth had tonsillitis with sore throat, fever, and vomiting on 8 July 1997. She was treated with penicillin for 10 days and improved. About 3 weeks later, she was taken to the emergency department of the local hospital with pain in both wrists and knees. She was found to be febrile, and throat culture subsequently grew Group A streptococci. She was given oral penicillin. In the following days, she developed migratory arthralgia and arthritis involving additional joints including her shoulders, elbows, wrists, and feet. She was seen at the IWK Grace Health Centre on 5 August for joint pain. A bone scan showed the presence of synovitis of multiple large and small joints. Treatment with an oral, non-steroidal anti-inflammatory agent was intitiated, and her joint symptoms improved within 24 hours. Investigation showed presence of elevated ESR (65 mm/hr), ASOT (> 1,000 IU/mL), and anti-DNaseB (1/680). ECG and echocardiogram were normal.

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Discussion

Acute rheumatic fever is a multi-system febrile disease affecting connective tissue of the heart and joints. It is now a rare disease in Canada. The case definition for acute rheumatic fever requires the presence of two major manifestations (carditis, migratory polyarthritis, chorea, erythema marginatum, and subcutaneous nodules) and one minor manifestation (fever, arthralgia, previous rheumatic fever, elevated ESR or C-reactive protein, and prolonged PR interval) in a subject with a documented recent streptococcal infection and no other explanation for the symptoms. The presence of one major and two minor manifestations also qualifies as a case of rheumatic fever. This is the first time in over 15 years that acute rheumatic fever has been seen in children in Nova Scotia. The first two cases came from the same geographic area, although they were unrelated and had no mutual contacts. The last case was from Yarmouth, a community 300 kilometers southwest of the other patients.

Since rheumatic fever is such an uncommon disease, the diagnosis may be missed[2]. However, periodic reports of outbreaks should help to remind physicians of this diagnosis[3]. When rash or arthritis symptoms occur after streptococcal pharyngitis or scarlet fever, physicians should be alerted to the possibility of acute rheumatic fever and investigate. Investigations to support the diagnosis should include throat culture for GAS and serologic testing to confirm a recent GAS infection. ESR is often significantly elevated out of proportion to the severity of the streptococcal infection. ECG and echocardiogram are important to confirm the presence or absence of carditis. Treatment with a non-steroidal anti-inflammatory agent such as aspirin typically results in rapid improvement of the arthritis. Long-term prophylaxis with penicillin is important to prevent future episodes of rheumatic fever and carditis. The drug of choice is benzathine penicillin 0.6 million units to 1.2 million units given once every 3 weeks.

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Source: *D Wong, MD, R Bortolussi, MD, B Lang, MD, Department of Pediatrics, IWK Grace Health Centre, Halifax, NS.*

EPIDEMIC METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS* STRAIN – ONTARIO

Several recent reports have indicated that the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in Ontario health-care facilities has increased significantly during the last few years[1-4]. In addition, these reports have suggested that this increase has been due primarily to the spread of a single MRSA strain. Since 1994, the Central Public Health Laboratory, Toronto, has received a steadily increasing number of *S. aureus* isolates reported as methicillin-resistant from hospitals and other institutions throughout the province. The number of MRSA isolates analyzed by the laboratory increased from 247 in 1994 to 354 in 1995, representing a 43% increase during this period. In 1996, a total of 1,418 MRSA isolates was analyzed; this represented a fourfold increase in the number of isolates analyzed over the previous year.

The number of MRSA isolates exhibiting phage type (PT) 95 increased markedly from 1994 to 1996 (Table 1). In 1994, PT 95 isolates represented only 3% of all MRSA isolates typed in the laboratory. In 1995, the frequency increased to 19%. In 1996, a total of 565 isolates were identified as PT 95, representing 40% of all MRSA isolates. This rapid increase in the frequency of MRSA PT 95 isolates was reflected in the increasing number of hospitals affected by these organisms between 1994 and 1996 (Table 1). In 1994, MRSA PT 95 isolates were encountered in only three hospitals in Ontario. However, the number of hospitals affected by these organisms increased sharply to 10 in 1995 and to 31 in 1996. In addition, MRSA PT 95 isolates were associated with outbreaks in several long-term care facilities during this period.

Table 1
MRSA isolates submitted for phage typing from Ontario health-care facilities

Year	No. of MRSA isolates (all types)	No. of MRSA isolates (PT 95)	No. of hospitals affected by MRSA (PT 95)
1994	247	7	3
1995	354	69	10
1996	1,418	565	31

Antimicrobial susceptibility testing in the Central Public Health Laboratory has shown that MRSA PT 95 isolates are uniformly resistant to several antimicrobial agents, including erythromycin, clindamycin, and ciprofloxacin; most isolates are also resistant to gentamicin and trimethoprim/sulphamethoxazole (Table 2). Resistance to vancomycin has not been observed among these isolates. The results of molecular typing have demonstrated that most MRSA PT 95 isolates have the same pulsed-field gel electrophoresis (PFGE) genome fragment pattern, but some have minor variations in their patterns which indicate that they represent subtypes of the predominant genotype[5]. Several unusual phenotypic properties, including a negative rapid-slide coagulase test, a negative or weak tube coagulase reaction at 4 hours but positive at 24 hours, and a negative or weak DNase reaction have been associated with isolates of this strain[6].

Table 2
Antimicrobial resistance patterns of MRSA PT 95 isolates from Ontario health-care facilities

Antimicrobial Agent	MRSA PT 95 (40 isolates)
Penicillin	R (40)
Cephalothin	R (40)
Erythromycin	R (40)
Clindamycin	R (40)
Ciprofloxacin	R (40)
Gentamicin	R (32)
TMP/SMX*	R (32)
Tetracycline	S (40)
Vancomycin	S (40)

R = Resistant
S = Susceptible
* = Trimethoprim/sulphamethoxazole

The recent emergence in Ontario of an epidemic MRSA strain exhibiting PT 95 has been preceded by the emergence of a similar epidemic strain in Europe. Since 1993, a multiply resistant MRSA strain with a genome fragment pattern corresponding to those of *S. aureus* strains of PT 95 has spread rapidly through health-care institutions in several regions of Germany[7,8]. Similarities between the PFGE patterns of the epidemic strains from Germany and Ontario suggest that these strains belong to the same clonal group of *S. aureus* strains exhibiting PT 95. Previous studies have indicated that PT 95 strains have a higher colonization capacity than strains of other phage types; this property may have played an important role in the rapid emergence of the epidemic strain in Ontario health-care facilities[9,10].

Acknowledgements

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Source: *M Preston, PhD, A Borczyk, MSc, F Jamieson, MD, Clinical and Environmental Bacteriology Department, Central Public Health Laboratory, Ministry of Health, Toronto, ON.*

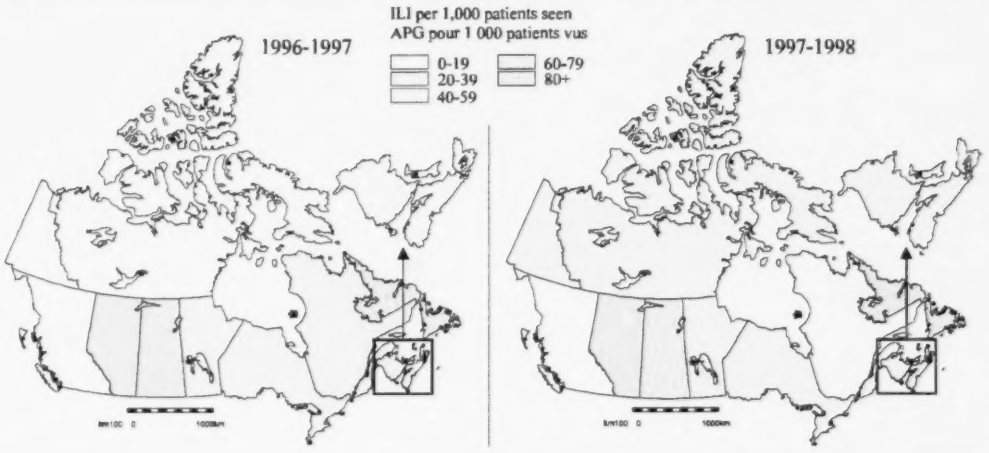
RESPIRATORY VIRUS SURVEILLANCE
FluWatch Project, 1997-1998

This update summarizes influenza activity until 20 February 1998. FluWatch has enrolled 208 sentinel physicians representing 139/288 (48%) census divisions in Canada. The physician response rate varies by province and by week. The mean response rate is 57% (42% to 68%). Figure 1 illustrates the standardized cumulative rates of influenza-like illness (ILI) by province for this and last season's FluWatch. Newfoundland and Alberta have the highest rates this season. Increases in the rate of ILI for 1997-1998 have been recorded in Newfoundland, Nova Scotia, Yukon, and Northwest Territories. The standardized rates of ILI reported to FluWatch (Figure 2) during the current season have shown an upward trend between weeks 4 and 8 (weeks ending 23 January and 20 February 1998, respectively). The highest rates of ILI, to date, have been in the < 10-year-old age groups (119 per 1,000 patients seen).

Since September 1997, the FluWatch program has received reports on 18,715 laboratory tests for influenza; 1,515 have been confirmed as influenza A and seven as influenza B. The provincial distribution of influenza A isolates which have not been subtyped is as follows: Nova Scotia (7), New Brunswick (31), Quebec (392), Ontario (748), Manitoba (47), Saskatchewan (17), and Alberta (200). Seventy-three influenza A isolates have been subtyped; 70 have been subtype H3N2 and three have been subtype H1N1. The provincial distribution of influenza A H3N2 is as follows: Saskatchewan (1), Alberta (2), and British Columbia (67). The three influenza A H1N1 cases are from Quebec. The provincial distribution of the seven influenza B isolates is as follows: Quebec (3) and Ontario (4).

Since November 1997, the National Laboratory for Viral and Zoonotic Pathogens, Laboratory Centre for Disease Control, has completed strain characterization on 41 influenza isolates as follows: 22 are A/Sydney/5/97 (H3N2)-like, seven are A/Texas/36/91 (H1N1)-like, and 12 are A/Wuhan/359/95 (H3N2)-like. As of 12 February, the Provincial Laboratory of British Columbia (PLBC), Vancouver, has completed strain identification on eight isolates, seven of which are A/Sydney/5/97-like. The provincial distribution of the 29 isolates characterized, to date, as A/Sydney-like is as follows: British

Figure 1
Standardized rates of ILI across Canada, by province, reported to FluWatch, 1 October 1996 to 20 February 1997 and 15 October 1997 to 20 February 1998



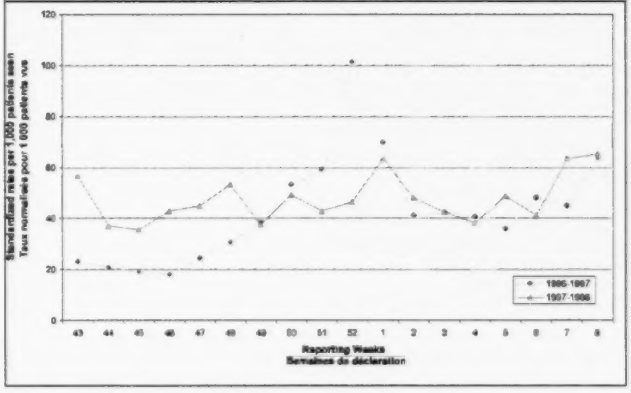
Columbia (7), Manitoba (1), Ontario (13), Quebec (7), and New Brunswick (1). All A/Texas isolates were from Ontario. The distribution of the A/Wuhan-like isolates is as follows: Ontario (2), Quebec (9), and Nova Scotia (1). The Provincial Laboratory of Public Health for Northern Alberta, Edmonton and the PLBC have each identified one isolate of A/Nanchang/933/95 (H3N2)-like. In summary, 50 influenza A isolates have been characterized: 29 (58%) are A/Sydney/5/97 (H3N2)-like, seven (14%) are A/Texas/36/91 (H1N1)-like, 12 (24%) are A/Wuhan/359/95 (H3N2)-like, and two (4%) are A/Nanchang/933/95 (H3N2)-like.

The A/Sydney/5/97-like strain, first identified in New Zealand and Australia in June 1997, is a related but antigenically distinguishable variant of the A/Wuhan/359/95-like virus strain. The A/Wuhan-like strain is an antigenically equivalent virus to the A/Nanchang/933/95-like strain included in the 1997-1998 influenza vaccine. Since vaccine effectiveness is dependent, in part, on the similarity between the strains included in the vaccine and the circulating strains, protection could be less than optimal in persons infected with A/Sydney-like viruses. Laboratory evidence suggests that vaccination with the currently available influenza vaccine will provide some protection against A/Sydney-like viruses. The A/Sydney-like strain was first isolated, in Canada, from two passengers aboard a cruise ship that sailed between New York and Montreal in September 1997. For the 1997-1998 influenza season, the A/Sydney/5/97-like strain has been isolated and characterized at the following WHO collaborating centres: Argentina, Australia, Austria, France, Hong Kong, Israel, Italy, Japan, Republic of Korea, Spain, Sweden, United Kingdom, and the United States.

As of 18 February 1998, international influenza activity continues to increase in the northern hemisphere. Influenza A has been the predominant influenza type. Where influenza A has been further identified, the H3N2 subtype was most frequently reported, except in the Russian Federation and the United Kingdom where H1N1 has been more common.

As of 20 February 1998 there were no new cases of influenza A (H5N1) ("bird flu" or "avian flu") in Hong Kong. The total number

Figure 2
Standardized rates of reported ILI across Canada, by week, reported to FluWatch, 15 October 1996 to 20 February 1997 and 15 October 1997 to 20 February 1998



of cases stands at 18 confirmed cases – all residents of Hong Kong Special Administrative Region. The onset date of illness of the last confirmed case was 28 December 1997. Two patients are still under treatment in hospital. Ten other patients have fully recovered and been discharged. Six people died of the disease.

FluWatch program reports can be accessed through the FluWatch Website:
http://www.hc-sc.gc.ca/hpb/lcdc/bid/dsd/fluwatch/index.html.
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Source: *P Buck, DVM, MSc, S Herman, C Scott, B Winchester, BSc, MSc, P Zabchuk, P Sockett, PhD, Chief, Division of Disease Surveillance, Bureau of Infectious Diseases, M Vanderkloot, BA, Bureau of Surveillance and Field Epidemiology, LCDC, Ottawa, ON.*

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- *Canadian contingency plan for viral hemorrhagic fevers and other related diseases* – Volume 23S1, January 1997.
- *An integrated protocol to manage health care workers exposed to bloodborne pathogens* – Volume 23S2, March 1997.
- *Infection control guidelines – Preventing the transmission of bloodborne pathogens in health care and public services settings* – Volume 23S3, May 1997.
- *Canadian national report on immunization*, 1996 – Volume 23S4, May 1997.
- *Canadian recommendations for the prevention and treatment of malaria among international travellers* – Volume 23S5, October 1997.
- *Proceedings of the national STD consensus meeting and national goals for the prevention and control of sexually transmitted diseases in Canada* – Volume 23S6, November 1997.
- *Controlling antimicrobial resistance: an integrated action plan for Canadians* – Volume 23S7, November 1997.
- *Infection control guidelines – Preventing the spread of vancomycin-resistant enterococci (VRE) in Canada; Foot care by health care providers; Preventing infections associated with indwelling intravascular access devices* – Volume 23S8, December 1997.
- *Notifiable diseases annual summary (1995)* – Volume 23S9, December 1997.

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